Amendments to the Specification

(1) Please replace the paragraph appearing at page 1, lines 9-11, with the following amended paragraph:

The present invention was made~~, at least in part,~~ with government support ~~funding received from the National Institutes of Health~~ under grant number RO1 DE11390 awarded by the National Institutes of Health. The U.S. government ~~may retain~~ has certain rights in this invention.

(2) Please replace the paragraph appearing at page 5, line 26 to page 7, line 2, with the following amended paragraph:

Megakaryocytes are the biggest cell of the bone marrow and the parent cell of platelets. Platelets are derived from the cytoplasm of megakaryocytes and are released to the bloodstream under the effects of cytokines such as IL-6 and IL-11 (Teramura et al., "Interleukin-11 Enhances Human Megakaryocytopoiesis *in vitro*," *Blood* 79:327-331 (1992); Burstein et al., "Thrombocytopoiesis in Normal and Sublethally Irradiated Dogs: Response to Human Interleukin-6," *Blood* 80:420-428 (1992)). Platelets are enucleate ~~nuclear~~ cells that have a plasma membrane, surface-connected canalicular and tubular system, mitochondria, granules, lysosomes, and peroxisomes (Bentfeld-Barker and Bainton, "Identification of Primary Lysosomes in Human Megakaryocytes and Platelets," *Blood* 59:472-481 (1982)). Recent studies demonstrate that platelets and many of their products are not only important in hemostasis, but have now emerged as important in immunoregulation and inflammation. For example, platelets produce key inflammatory mediators such as transforming growth factor-β (TGF-β), thromboxane A_2, and PGE_2 (Scheuerer et al., "The CXC-chemokine Platelet Factor 4 Promotes Monocyte Survival and Induces Monocyte Differentiation into Macrophages," *Blood* 95:1158-1166 (2000); Gear et al., "Platelet Chemokines and Chemokine Receptors: Linking Hemostasis, Inflammation, and Host Defense," *Microcirculation* 10:335-350 (2003); Vezza et al., "Prostaglandin E2 Potentiates Platelet Aggregation by Priming Protein Kinase C," *Blood* 82:2704-2713 (1993)). The recent key demonstration that activated human platelets express and expel CD40 ligand (CD40L, formally known as CD154) provides a mechanism of interaction with CD40 expressing cells that include macrophages and vascular structural cells (Phipps, "Atherosclerosis: The Emerging Role of Inflammation and the CD40-CD40 Ligand System," *Proc. Natl. Acad. Sci. USA* 97:6930-6932 (2000); Phipps et al., "Platelet Derived CD154 (CD40 Ligand) and Febrile Responses to Transfusion," *Lancet*

357:2023-2024 (2001); Danese et al., "Platelets Trigger a CD40-Dependent Inflammatory Response in the Microvasculature of Inflammatory Bowel Disease Patients," *Gastroenterology* 124:1249-1264 (2003); Henn et al., "CD40 Ligand on Activated Platelets Triggers an Inflammatory Reaction of Endothelial Cells," *Nature* 391:591-594 (1998)). These cells when activated through CD40 express Cox-2 and prostaglandins, adhesion molecules, and cytokines such as IL-6 and tissue factor (Mach et al., "CD40 Signaling in Vascular Cells: A Key Role in Atherosclerosis?" *Atherosclerosis* 137:S89-95 (1998); Linton and Fazio, "Cyclooxygenase-2 and Atherosclerosis," *Curr. Opin. Lipidology* 13:497-504 (2002)). Many new studies now demonstrate that elevated CD40L levels in blood are associated with acute coronary syndromes and stroke (Heeschen et al., "Soluble CD40L in Acute Coronary Syndromes," *New Engl. J. Medicine* 348:1104-1111 (2003)). Interestingly, elevated serum levels of CD40L predict an increased cardiovascular risk in a healthy population (Schonbeck et al., "Soluble CD40L and Cardiovascular Risk in Women," *Circulation*. 104:2266-2268 (2001)).